Response to Comments on Final Public Review Draft Prioritization of Toxic Air Contaminants Under the Children's Environmental Health Protection Act

July, 2001

Responses to Comments on Diesel Particulate Submitted by American Trucking Association

Comment 1: OEHHA has not Provided Interested Parties with Adequate Time in which to Comment on the Revised Listing.

As a preliminary matter, we note that notice concerning OEHHA's decision to revise the prioritization of TACs under the Children's Environmental Health Protection Act was not posted on OEHHA's website until June 29, 2001, leaving stakeholders with a very generous comment period of 2 weeks (which includes 2 weekends and the Fourth of July holiday). This abbreviated comment period is inadequate to prepare comprehensive comments on such a complex subject. As a result, ATA and other interested stakeholders have been denied a reasonable opportunity to offer detailed comments on the decision to replace benzene and formaldehyde with diesel exhaust particulate and acrolein as Tier 1 substances under the Children's Environmental Health Protection Act.

ATA recognizes that the statutory deadline for this listing passed on July 1, 2001; however, the reshuffling of listed chemicals immediately prior to the statutory deadline without an adequate opportunity to receive meaningful public comment is a clear violation of administrative procedure that will not withstand judicial scrutiny.[1] To cure this procedural defect, OEHHA should publish a formal extension to the comment period and allow interested parties an opportunity to supplement the record.

Response 1: The comment indicates that there was little time to comment on the revised prioritization. It must be noted that the same arguments that diesel exhaust particulate may cause infants and children to be especially susceptible to illness were made in the initial and latest versions of the prioritization document. In the initial and later versions, it was made clear in the text of the document that the prioritization into the top Tier of 5 TACs for listing under Health and Safety Code Section 39669.5 was subject to public and peer review comments. Thus, interested parties knew that it was quite possible any of the chemicals discussed in the document could end up in the final five proposed for listing under SB 25. The initial draft of the prioritization document was posted on March 7th for a 30-day comment period (ATA provided comments then). The document was open for comment during the Panel deliberations (which started officially at the first SRP meeting on the subject held on April 27th). At the Scientific Review Panel Meeting June 15th, the SRP provisionally approved OEHHA's suggested revisions to the prioritization document and Tiers 1 and 2 pending a final public comment period on the document. We discussed in the record when that public comment period would start and end. In attendance at that public meeting was legal representation for the Engine Manufacturers Association, an organization with which ATA is in close communication. Furthermore, the revised

[1] *See* Cal. Gov't Code Sections 11346.4, 11346.8.

document was posted on our webpage along with the notice of the public comment period on June 23rd. Thus, there was a longer period of time to develop comments than the comment indicates. Furthermore, no new lines of evidence were brought forth in the final document as regards diesel exhaust particulate and potential differential impacts on infants and children, and no new issues are being raised by ATA; therefore, it is unclear why the commenter needs more time.

As the comment notes, there was a very tight timeline for this entire process.

Comment 2: OEHHA Should Delay Listing Diesel Exhaust Particulate Matter as a Tier 1 Substance in Light of Pending Litigation.

In our initial comments, we noted that the listing of diesel exhaust as a TAC in the State of California currently is the subject of litigation.[2] This litigation questions whether the classification of diesel exhaust particulate matter as a TAC is supported adequately by scientific evidence. The legal challenge to the diesel exhaust particulate matter TAC listing already has withstood the government's demurrer, with Judge Stephen Kane having ruled that the suit alleges valid causes of action. The case now will be heard on the merits. Accordingly, it is inappropriate to list diesel particulate as a Tier 1 substance until the litigation concludes.

Response 2: There is no reason to delay listing under SB 25. Diesel exhaust particulate is an identified toxic air contaminant and therefore subject to consideration for listing under SB 25.

Comment 3: OEHHA'S Basis for Listing Diesel Particulate Matter in Tier 1 is Unsupported by the Scientific Evidence in the Record.

OEHHA erroneously included diesel exhaust among the five Tier 1 TACs that disproportionately impact children. OEHHA sets forth the following reasons for including diesel exhaust particulate in Tier 1:

[D]iesel exhaust particulate is ubiquitous in urban environments, and exposures are widespread. There are many studies demonstrating that diesel exhaust particulate can enhance allergic responses, and induce new allergies to airborne allergens. This raises concern for enhancement of allergic airway

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[2] *See Apodaca v. California Air Resources Board*, (Superior Court Case No. 00CECG10832).

disease including asthma, and for development of new asthma. Diesel exhaust particles contribute to ambient PM10. Ambient PM10 has been shown to exacerbate asthma and has been associated with low birth weight and decreased lung function in children. In addition, diesel exhaust particulate contains PAHs [polycyclic aromatic hydrocarbons] (and other mutagenic polycyclic organic matter).[3]

The above paragraph indicates that OEHHA's decision to elevate diesel exhaust particulate matter to Tier 1 is based upon the following three observations:

Diesel exhaust particles contribute to ambient PM_{10};
Diesel exhaust particles contain PAH; and
Diesel exhaust particulate may exacerbate asthma and enhance allergic responses.

As demonstrated in these comments and the initial comments submitted by the ATA, these observations and the inconclusive science they are based upon are insufficient to support a conclusion that diesel exhaust "may cause children and infants to be especially susceptible to illness."

Comment 3a: Diesel Exhaust is a small contributor to ambient particulate matter.

OEHHA's statement that diesel exhaust particulate is ubiquitous in urban environments and contributes to ambient particulate matter, overstates the impact diesel exhaust has upon ambient particulate matter. Diesel exhaust is a very small component of PM_{10}. Recent EPA national assessment data reports diesel exhaust (from both on-road and non-road sources) contributed only 1.3% of total emitted PM_{10} and 4.9% of total emitted $PM_{2.5}$. Thus, it is misleading to characterize diesel exhaust as a significant source of ambient particulate matter.

Even if one could completely eliminate diesel exhaust from the ambient air, the impact of particulate matter upon infants and children likely would remain. As such, control of diesel exhaust as a separate air contaminant will not solve the problem of exposure to ambient particulate matter, which already receives adequate regulatory attention as a criteria pollutant for which levels are set to protect the health of sensitive populations such as infants and children. OEHHA should focus its attention on air toxics for which additional regulation will result in tangible health benefits for infants and children.

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[3] Office of Environmental Health Hazard Assessment California Protection Agency "Prioritization of Toxic Air Contaminants Under the Children's Environment Health Protection Act" Final Public Review Draft, p. 35 (June 2001).

Response 3a: As stated in the diesel exhaust SB25 prioritization data summary document, based on the ARB 1990 emissions inventory, approximately 58,000 tons of diesel exhaust PM_{10} from all sources are emitted into California air each year (ARB, 1997). ARB staff have estimated that emissions from diesel exhaust contribute about 3 and 8 percent of the total California PM_{10} and $PM_{2.5}$ inventories, respectively (ARB, 1997). This is averaged over all sources and therefore is not reflective of the percentage of PM_{10} or $_{2.5}$ that is diesel exhaust particulate in areas with heavy traffic (e.g., near freeways and in highly urbanized areas). The statewide population-weighted average diesel exhaust PM concentration is estimated to be 3.2 $\mu g/m^3$. An ARB study to determine the PM_{10} concentrations due to the primary emissions from diesel engine exhaust near the Long Beach Freeway indicated that near-roadway concentrations of diesel exhaust PM_{10} may be as high as 8 $\mu g/m^3$ above ambient concentrations for one 24-hour period (ARB, 1996). This is notable in light of the fact that the chronic Reference Exposure Level (REL) for diesel exhaust is 5 $\mu g/m^3$. In addition, measurements conducted by ARB in vehicles on Los Angeles freeways indicated concentrations of black soot up to 23 $\mu g/m^3$, which were strongly influenced by presence of diesel vehicles in front of the test vehicle. After considering these facts along with the cancer and noncancer health effects data for diesel exhaust, it is clear that diesel exhaust is in fact an important source of air pollutants. The exposure assessment section of the diesel TAC identification document also observes:

> "These total exposures estimates are believed to underestimate, to an unknown extent, Californians' actual exposures to diesel exhaust particles. This is because insufficient data are available for concentrations inside vehicles and along roadways to allow such near-source, elevated exposures to be estimated for the population". (ARB, 1998, page A-57)

Zelinska (1991, cited by ARB, 1998) found that motor vehicle exhaust was the second highest contributor to wintertime PM_{10}, and that diesel-fueled motor vehicle exhaust was responsible for at least half of the motor vehicle derived PM_{10}. Also, as described by ARB (1998), PAHs are generally associated with the particles composed of elemental carbon (EC), rather than the mineral particles of geological or atmospheric origin. ARB found that

> "… diesel emissions were responsible for approximately 67 percent of the fine EC mass in the Los Angeles atmosphere, and that the exhaust particles averaged about 64 percent EC." (ARB, 1998, page A-47)

The document does not state that diesel exhaust is a significant source of PM for the entire state of California. It does state that "Although the contribution of diesel exhaust particulate to the statewide average PM_{10} is relatively small in California (5% or so), it is a more significant portion of PM_{10} and $PM_{2.5}$ in urban locations". It should also be noted that diesel exhaust particulate causes adverse immune system effects, which may result in

adverse health outcomes (e.g. possible exacerbation of asthma and allergic rhinitis) (Diaz-Sanchez *et al*., 2000), that are not shared by other model particulates such as carbon black and crystalline silica (van Zijverden et al., 2000). This suggests that diesel exhaust particulate has additional unique toxicological properties above and beyond the cardiopulmonary morbidity and mortality associated with exposure to general ambient PM_{10}.

Comment 3b: Diesel Exhaust Particulate's Polycyclic Aromatic Hydrocarbon Content does Not Justify the Listing of Diesel Exhaust as Potentially Having a Disproportionate Impact Upon Children.

OEHHA reasons that "diesel exhaust particulate contains PAHs." In fact, OEHHA has not actually quantified the contribution of diesel exhaust particulate matter to ambient PAH. More importantly, OEHHA will list PAHs independently among the Tier 1 substances. Therefore, to the extent that PAHs contained in diesel exhaust may cause children and infants to be especially susceptible to illness, these impacts would be addressed following the finalization of PAHs on the Tier 1 list. A separate listing for diesel exhaust particulate matter based partly upon the presence of PAHs is unwarranted.

As highlighted in our original comments, OEHHA has not investigated whether the diesel exhaust particles bind or release PAHs in the presence of bodily fluids. Prior to listing diesel exhaust as a contaminant that may disproportionately impact the health of children based upon its PAH content, it is important to know whether PAHs are absorbed into the body. It is theoretically possible for PAHs to remain bound to diesel exhaust particulates, thereby having no adverse health effect once absorbed into the body. OEHHA cites to no studies attempting to describe how the body metabolizes diesel exhaust. In the absence of such evidence, the mere presence of PAHs in diesel exhaust is insufficient to form a conclusion of adverse health effects.

Response 3b: It is well documented that all the physical phases of diesel exhaust contain PAHs, and contribute to ambient PAHs. An exact quantification of the contribution of diesel exhaust particulate matter to total ambient PAH is not a necessary requirement for listing diesel exhaust in Tier I. The bioavailability of PAHs contained in diesel exhaust was thoroughly reviewed in the diesel exhaust TAC document (OEHHA, 1998). The studies reviewed clearly indicated that the PAHs in diesel exhaust were bioavailable upon inhalation exposure. Additionally, a recent study by Sato *et al.* (2000) indicated that rats exposed to diesel exhaust by inhalation demonstrated increased mutations in a reporter gene and covalent DNA adducts, additional evidence suggesting PAH bioavailability. However, the presence of PAHs in diesel exhaust was not the sole reason for the listing of diesel exhaust in Tier I of the prioritization document.

Mechanistic data indicates that diesel exhaust particulate matter may exert specific effects on the immune system that are not shared by some other PM components such as crystalline silica (van Zijverden *et al.*, 2000). Additionally, diesel exhaust has immune system effects not necessarily shared by other model particulates such as carbon black (Diaz-Sanchez *et al.*, 2000). Some of these effects, such as enhancing ovalbumin-induced IgG1 and IgE levels in mice were also shown by extracts of DEPM and the polycyclic aromatic hydrocarbons (PAHs) phenanthrene and anthracene (Heo *et al.*, 2001). However, these same effects were not shown by the PAHs 3-methylcholanthrene and acenaphthylene or by 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD), suggesting that the effects may not be globally attributable to either all PAHs or *Ah* receptor ligands. This indicates that non-PAH components of diesel exhaust could potentially also be exerting adverse immune system effects, in addition to other adverse health effects associated with PM exposure.

Comment 3c: Diesel exhaust particulate can exacerbate asthma and enhance allergic responses.

As stated in our original comments, OEHHA states that diesel exhaust contributes to ambient particulate matter and then notes that ambient particulate matter has been shown to exacerbate asthma and has been associated with low birth weight and decreased lung function. Based upon this evidence, OEHHA concludes that diesel exhaust, as a component of particulate matter, is a substance of concern. However, OEHHA cites no scientific evidence directly linking diesel exhaust to these adverse effects or demonstrating that exposure to diesel exhaust triggers an adverse health effect different from exposure to particulate matter generally.[4]

The available studies that measure the effect on children of diesel exhaust exposure are limited, both in number and in design. The studies that measure the non-cancerous effects of diesel exhaust exposure in adults, however, consistently show no effect or only acute transient effect on respiratory status. The available studies, therefore, do not provide a basis to conclude that diesel particulate matter places children at a greater risk for non-cancerous respiratory illness. Additionally, the ambient particulate matter levels that may contribute to respiratory illnesses are comprised of various emission sources, of which diesel exhaust is a small fraction. Current regulations have reduced, and will continue to reduce diesel exhaust emissions.

Response 3c: There are a number of studies demonstrating adverse health impacts in infants and children from exposure to PM_{10} in ambient air. The adverse health outcomes measured range from exacerbation of asthma symptoms to infant and child mortality.

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[4] *See* ATA's Initial Comments, Section III.B.2.a, attached hereto as Appendix A.

These studies took place in a number of cities with varying percentage of total PM_{10} that is diesel exhaust particulate. However, in some of the cities studied, diesel exhaust particulate contributes the vast majority of PM_{10}. There was certainly no protective effect of diesel exhaust particulate in these studies. It is therefore reasonable to conclude that diesel exhaust particulate as a component of airborne PM_{10} has the adverse effects attributable to airborne PM_{10}.

There are several dozen studies demonstrating enhancement of allergic responses in both humans and animals following exposure to diesel exhaust particulate. As noted in the response to Comment 3b, diesel exhaust particulate causes adverse immune system effects that may result in adverse health outcomes (e.g. possible exacerbation of asthma and allergic rhinitis) (Diaz-Sanchez *et al*., 2000, and others; see diesel exhaust summary pp. 7-13); these adverse immunological effects are not shared by other model particulates such as carbon black and crystalline silica (van Zijverden *et al*., 2000). This suggests that diesel exhaust exhibits noncancer health effects that are unique over and above the cardiopulmonary morbidity and mortality associated with exposure to ambient general particulate matter. Acute exposures of healthy adult humans to concentrations of diesel exhaust particulate matter (300 $\mu g/m^3$) approximately one order of magnitude greater than peak diesel exhaust concentrations noted near California freeways demonstrated a marked leukocytic airway infiltration accompanied by enhanced chemokine and cytokine production (Salvi *et al.*, 2000). It should be noted that 300 $\mu g/m^3$ was a LOEL (Lowest Observable Effect Level) in this study. Lower concentrations of diesel exhaust particulate matter were not tested, raising the possibility that these effects may be observed at concentrations lower than 300 $\mu g/m^3$. Additionally, Frew *et al.* (2001) observed upregulation of IL-10 production in the bronchial epithelium of asthmatic subjects but not healthy subjects at a PM_{10} concentration of only 108 $\mu g/m^3$. The authors stated that the observed IL-10 upregulation may alter the airway biology towards a more allergic phenotype. It is also possible that healthy and/or asthmatic children may be more sensitive to diesel exhaust particulate matter-induced immune system effects than healthy adults. These data indicate that diesel exhaust particulate matter adversely impacts healthy and asthmatic adult immune systems at concentrations close to those observed in cars driving on California freeways (e.g., about 25 $\mu g/m^3$), making them very relevant to a consideration of diesel exhaust particulate matter for prioritization under SB 25.

Comment 3d: **Replacing Diesel Exhaust Particulate in Place of Benzene and Formaldehyde is Contrary to the Statutory Listing Criteria Under the Children's Environmental Health Protection Act.**

The last minute substitution of diesel particulate matter in place of a substance such as benzene defies logic and cannot be justified under the statutory criteria established by the Children's Environmental Health Protection Act. Most significantly,

benzene is a known carcinogen, whereas diesel particulate is suspected of aggravating respiratory conditions such as asthma.

No scientific studies substantiate OEHHA's conclusion that diesel exhaust particulate enhances allergic responses, induces new allergies to airborne allergens, and may enhance allergic airway disease. The published literature on diesel particulate matter suggest that at high concentrations, the PAHs adsorbed to diesel particulate matter *may* act as an adjuvant to enhance allergic responses in people already pre-sensitized to an allergen when co-administered with an allergen. There is no, and the OEHHA does not cite to, scientific evidence that diesel exhaust particulate enhances allergic airway disease. Available studies performed on volunteers directly exposed to diesel exhaust under experimental laboratory conditions show at most only transient effects of diesel exhaust on respiratory function. There are no human exposure or epidemiological studies that show diesel exhaust exposure causes chronic respiratory disease.

The most recent studies characterize the soluble organic chemical portion of diesel particulate matter as the adjuvant, not the actual carbon particle. Exposure to carbon particle alone in a murine model induced no immuno-adjuvant effect. The soluble organic chemical agents on the carbon black particle include PAHs and other chemicals. As stated previously, PAHs already are listed as a Tier 1 pollutant. It therefore makes no logical sense to remove benzene from Tier 1 in order to elevate the importance of diesel particulate, when the scientific evidence suggests the only component of diesel exhaust particulate that causes any appreciable health effect is already addressed as a Tier 1 pollutant.

Response 3d: Diesel exhaust particulate has been one of the candidates for listing under SB 25 since the beginning of this process and was listed as a candidate in the initial draft of the document. Thus, the comment's statement that diesel exhaust particulate was a last minute substitution for benzene is a bit misleading. There were 12 top candidates in the initial draft of the document which clearly stated in several places that the placement of the chemicals into two Tiers was subject to public and peer review comment. In addition, the first paragraph of the comment indicates that while benzene is a carcinogen, diesel exhaust particulate only exacerbates asthma. The Toxic Air Contaminant (TAC) document for Particulate Matter From Diesel-Fueled Engines (OEHHA, 1999) discussed in detail the more than 3 dozen studies showing elevated risks for lung cancer in diesel exhaust exposed workers. The TAC Identification document for diesel describes the development of a cancer unit risk value range for diesel exhaust, and states "a reasonable and likely explanation for the increased risks of lung cancer observed in the epidemiologic studies is a causal association between diesel exhaust exposure and lung cancer". Diesel exhaust exposure carries a cancer risk in addition to non-cancer toxic effects, including possible exacerbation of asthma. Diesel exhaust is also regulated as a carcinogen by the Occupational Safety and Health Administration; the International Agency for Research on Cancer classifies diesel exhaust as a 2A carcinogen.

There are several dozen studies which provide evidence that diesel exhaust particulate enhances allergic responses to allergens and even to neo allergens in both humans and animals (see Diesel exhaust particulate summary, pp.7-13). As noted in the responses to Comments 3a, b and c, diesel exhaust particulate demonstrates immune system effects (which may result in adverse health outcomes such as exacerbation of asthma and allergic rhinitis) (Diaz-Sanchez *et al*., 2000) that are not shared by other model particulates such as carbon black and crystalline silica (van Zijverden *et al*., 2000). This suggests that diesel exhaust exhibits noncancer health effects that are unique over and above the cardiopulmonary toxic effects of exposure to ambient general particulate matter. Some of these effects, such as enhancing ovalbumin-induced IgG1 and IgE levels in mice were also shown by extracts of DEPM and the polycyclic aromatic hydrocarbons (PAHs) phenanthrene and anthracene (Heo *et al.*, 2001). However, these same effects were not shown by the PAHs 3-methylcholanthrene and acenaphthylene or by 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD), suggesting that the effects may not be globally attributable to either all PAHs or *Ah* receptor ligands. This indicates that non-PAH components of diesel exhaust could potentially also be exerting adverse immune system effects, in addition to other adverse health effects associated with PM exposure.

Acute healthy adult human exposures to concentrations of diesel exhaust particulate matter (300 $\mu g/m^3$) approximately one order of magnitude greater than peak diesel exhaust concentrations noted near California freeways demonstrated a marked leukocytic airway infiltration accompanied by enhanced chemokine and cytokine production (Salvi *et al.*, 2000). This represents an allergic inflammatory reaction. It should be noted that 300 $\mu g/m^3$ was a LOEL (Lowest Observable Effect Level) in this study. Lower concentrations of diesel exhaust particulate matter were not tested, raising the possibility that these effects may be observed at concentrations lower than 300 $\mu g/m^3$. Additionally, Frew *et al.* (2001) observed upregulation of IL-10 production in the bronchial epithelium of asthmatic subjects but not healthy subjects at a PM_{10} concentration of only 108 $\mu g/m^3$. The authors stated that the observed IL-10 upregulation may alter the airway biology towards a more allergic phenotype. It is also possible that healthy and/or asthmatic children may be more sensitive to diesel exhaust particulate matter-induced immune system effects than healthy adults. These data indicate that diesel exhaust particulate matter adversely impacts healthy adult immune systems at concentrations close to those observed near California freeways, making them very relevant to a consideration of diesel exhaust particulate matter for prioritization under SB 25.

The comment also appears to imply that the only toxic constituent of diesel exhaust particles are the PAHs which may be actors in the observed enhancement of allergy in humans and animals. While there is a line of evidence that indicates PAHs are involved in this response, it is by no means clear that they are the only chemicals absorbed to the carbon core that influence the allergic inflammatory responses observed.

OEHHA has also noted in the prioritization document and in its response to comments that no direct epidemiological evidence of differential sensitivity of children to asthma induced specifically by diesel exhaust particulate matter (as opposed to PM_{10} or $PM_{2.5}$) has been published. OEHHA considers asthma to impact children more than adults primarily because children have higher prevalence rates for asthma and are hospitalized more often than adults for asthma. The possibility that diesel exhaust particulate matter may differentially impact children stems from the mechanistic data indicating that diesel exhaust particulate matter exerts specific adverse immune system effects. The adverse health effects observed in the traffic studies cited in the document, as well as the epidemiological studies observing a positive correlation between PM_{10} and exacerbation of asthma, provide additional support for the conclusion that diesel exhaust particulate may exacerbate asthma.